

The opinion in support of the decision being entered today was not written
for publication and is not binding precedent of the Board.

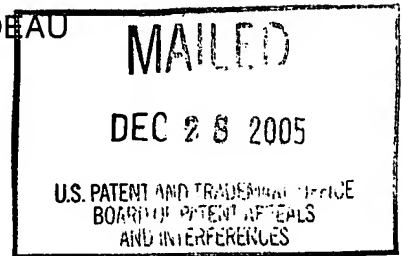
UNITED STATES PATENT AND TRADEMARK OFFICE

**BEFORE THE BOARD OF PATENT APPEALS
AND INTERFERENCES**

Ex parte BRUCE A. YANKER and PHILIP NADÉAU

Appeal No. 2005-2028
Application No. 10/086,398

HEARD: November 15, 2005



Before ELLIS, SCHEINER and ADAMS, Administrative Patent Judges.

SCHEINER, Administrative Patent Judge.

DECISION ON APPEAL

This is a decision on appeal under 35 U.S.C. § 134 from the final rejection of
claims 23-29, the only claims remaining in the application.

BACKGROUND

"Alzheimer's disease (AD) is the most common cause of dementia in the aged
population. The accumulation of large numbers of senile plaques containing the 40-42
amino acid amyloid β protein ($A\beta$) is a classic pathological feature of AD. Both genetic
and cell biological findings suggest that the accumulation of $A\beta$ in the brain is the likely
cause of AD" and "it is widely believed that drugs which decrease the levels of $A\beta$ in the
brain would prevent Alzheimer's disease." Specification, page 1. "Blood cholesterol
levels are correlated with production of amyloid β protein ($A\beta$)," thus, according to
appellants, "[m]ethods for lowering blood cholesterol levels can be used to decrease
production of $A\beta$, thereby decreasing the risk of developing AD." *Id.*, page 2.

Further according to appellants, “lower[ing] cholesterol blood levels at least 10% . . . is believed to be sufficient to decrease production of A β .” Id., page 3. “Methods include administration of compounds which increase uptake of cholesterol by the liver, . . . compounds which block endogenous cholesterol production, . . . compositions which prevent uptake of dietary cholesterol, and administration of combinations of any of these which are effective to lower blood cholesterol levels.” Id., pages 2-3. “HMG CoA reductase inhibitors have been shown to lower blood cholesterol levels by upregulating lipoprotein clearance receptors in the liver” and by “directly inhibit[ing] cholesterol synthesis in neurons.” Id., page 6. “Representative CoA reductase inhibitors include . . . lovastatin, simvastatin, compactin, fluvastatin, atorvastatin, cerivastatin, and pravastatin.” Id. “Compounds which inhibit cholesterol biosynthetic enzymes[] includ[e] [those which inhibit] 2,3-oxidosqualene cyclase, squalene synthase, and 7-dehydrocholesterol reductase” (id.). Finally, bile acid binding resins, fibrates, probucol, nicotinic acid, garlic and garlic derivatives, and psyllium “are also used to lower blood cholesterol levels” (id.).

The claims on appeal are directed to compositions for decreasing the production of A β , and read as follows:

23. A composition for decreasing the production of A β comprising an effective amount of a compound decreasing blood cholesterol levels to decrease A β production by neuronal cells in an individual at risk of developing Alzheimers.

24. The composition of claim 23 comprising an HMG CoA reductase inhibitor.

25. The composition of claim 24 wherein the inhibitor is selected from the group consisting of lovastatin, simvastatin, fluvastatin, pravastatin, atorvastatin, cerivastatin, and compactin.

26. The composition of claim 23 comprising a compound which inhibits uptake of dietary cholesterol.

27. The composition of claim 23 wherein the composition blocks or decreases endogenous cholesterol production.

28. The composition of claim 27 wherein the composition comprises an inhibitor of the cholesterol biosynthetic enzymes selected from the group consisting of 2,3-oxidosqualene cyclase, squalene synthase, and 7-dehydrocholesterol reductase.

29. The composition of claim 23 wherein the composition is selected from the group consisting of a fibrate, a bile acid binding resin, probucol, nicotinic acid, garlic or garlic derivative, and psyllium.

The references relied on by the examiner are:

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|--------------------------------|-----------|---------------|
| Hoffman et al. (Hoffman) | 4,866,090 | Sep. 12, 1989 |
| Wannamaker et al. (Wannamaker) | 5,350,758 | Sep. 27, 1994 |
| Spielvogel et al. (Spielvogel) | 5,362,732 | Nov. 8, 1994 |

The claims stand rejected as follows:

- I. Claims 23-27 under 35 U.S.C. § 102(b) as anticipated by Hoffman.
- II. Claims 23, 27 and 28 under 35 U.S.C. § 102(b) as anticipated by Wannamaker.
- III. Claims 23 and 29 under 35 U.S.C. § 102(b) as anticipated by Spielvogel.
- IV. Claims 26-27 under 35 U.S.C. § 103(a) as unpatentable over Wannamaker.

We affirm the rejections under 35 U.S.C. § 102(b) and do not reach the rejection under 35 U.S.C. § 103(a).

DISCUSSION

Hoffman describes lovastatin, pravastatin and simvastatin, "HMG-CoA reductase inhibitors [which] are useful as anti-hypercholesterolemic agents" (Hoffman, column 2, lines 34-39), and which may be administered in a "daily dosage . . . of from about 10 mg to 2000 mg" (*id.*, column 9, lines 58-60).

Wannamaker describes piperidyl sulfonamides and piperidyl sulfoxamides which "inhibit cholesterol biosynthesis and are useful in lowering blood cholesterol" by inhibiting squalene epoxidase and/or oxidosqualene cyclase. Wannamaker, column 2, lines 12-15.

Finally, Spielvogel describes nicotinic acid and probucol, “known hypolipidemic agents” (Spielvogel, column 8, lines 38-40).

We agree with the examiner that the subject matter of claims 23-27 is anticipated by Hoffman; the subject matter of claims 23, 27 and 28 is anticipated by Wannamaker; and the subject matter of claims 23 and 29 is anticipated by Spielvogel.

There is no dispute that the anti-hypercholesterolemia agents described in the references relied on by the examiner are the same as the anti-hypercholesterolemia agents required by the claims. Rather, appellants argue that “[t]here is no teaching or suggestion in the prior art that the compounds disclosed have any effect on the production of A β protein in neuronal cells.” Brief, page 7. In addition, appellants assert that “dosages that are effective in lowering the amount of amyloid precursor protein to decrease production of A β are different from those [effective in] lowering cholesterol to treat or prevent atherosclerosis” (*id.*), since “a 10% decrease in serum cholesterol levels is believed to be sufficient to decrease production of A β protein in neurons” (*id.*), which, according to appellants, “would not be clinically effective in treating hypercholesterolemia” (*id.*).

With respect to appellants’ first argument, it is well settled that a compound known in the prior art is not rendered patentable by a recitation of properties, whether or not the properties are shown or suggested in the prior art. In re Spada, 911 F.2d 705, 709, 15 USPQ2d 1655, 1658 (Fed. Cir. 1990). That is, a prior art reference may anticipate even when claim limitations are not expressly found in that reference, but are nonetheless inherent in it. See, e.g., Atlas Powder Co. v. IRECO Inc., 190 F.3d 1342, 51 USPQ2d 1943 (Fed. Cir. 1999); Titanium Metals Corp. v. Banner, 778 F.2d 775, 227 USPQ 773 (Fed. Cir. 1985). Appellant’s burden is not discharged by including

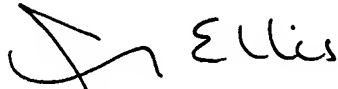
additional and assertedly different properties in the claims. A chemical compound and its properties are inseparable. See In re Papesch, 315 F.2d 381, 391, 137 USPQ 43, 51 (CCPA 1963). Again, when a compound is not novel it is not rendered patentable by a recitation of properties, whether or not these properties are shown or suggested in the prior art. See In re Spada, 911 F.2d at 709, 15 USPQ2d at 1658.

With respect to appellants' second argument, the underlying premise appears to be that a lesser amount of a known compound is somehow patentable over a larger amount of the same compound - even when there is no change in the structure or formulation of the compound. Appellants do not rely on any authority for this premise, nor do we know of any. In any case, the claims are not limited to an amount "effective in lowering the amount of amyloid precursor protein to decrease production of A β . . . [but not effective in] lowering cholesterol to treat or prevent atherosclerosis" (Brief, page 7), nor is there any evidence to suggest that an amount high enough to lower serum cholesterol levels would not, at the same time, lead to decreased production of A β .

The rejection of claims 23-27 as anticipated by Hoffman is affirmed; the rejection of claims 23, 27 and 28 as anticipated by Wannamaker is affirmed; and the rejection of claims 23 and 29 as anticipated by Spielvogel is affirmed. Our affirmance of these rejections constitutes a disposition of this appeal, and we find it unnecessary to reach the rejection of claims 23-27 under 35 U.S.C. § 103(a) as unpatentable over Wannamaker.

No time period for taking any subsequent action in connection with this appeal
may be extended under 37 CFR § 1.136(a).

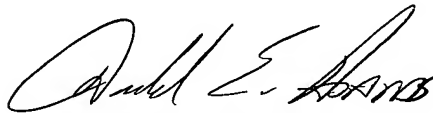
AFFIRMED



Joan Ellis
Administrative Patent Judge



Toni R. Scheiner
Administrative Patent Judge



Donald E. Adams
Administrative Patent Judge

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